With the author's

compliments.

THREE LECTURES ON EPILEPSY

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ON

EPILEPSY

BEING THE MORISON LECTURES DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH IN 1910

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PREFACE

The following three Lectures, being the Morison Lectures for 1910, were delivered before the Royal College of Physicians of Edinburgh. They have been already published with some abbreviations in the *British Medical Journal* for March and April 1910. They are reprinted here *in extenso*.

It is a pleasant duty to express my cordial thanks to Dr. Alexander Morison and to the President and Council of the Royal College of Physicians for giving me an opportunity of briefly, though I fear imperfectly, stating the present views upon an old but ever interesting disease.

18 Harley Street, London,
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THREE LECTURES ON EPILEPSY

LECTURE I.

THE PROBLEM OF EPILEPSY.

Mr. President and Fellows of the College,—My first and most agreeable duty on this occasion is to express to you, Sir, and the Council of the College, my thanks for conferring upon me the honour of delivering the Morison Lectures for the current year. I regard it as an especial favour, for amongst my predecessors in this lectureship, few only have not been Fellows of this College.

Although the lecturer was primarily intended to give a discourse upon some subject bearing upon mental disease, the scope has during recent years been enlarged so as to permit reference to diseases of the nervous system and allied subjects.

In selecting Epilepsy, therefore, as the theme of these lectures, I have chosen a disease which exhibits both psychical and neurological features.

Historical.—Epilepsy is a malady which has aroused popular as well as medical interest at all times. To the medical mind, it is of interest both on account of the frequency of its occurrence, the difficulty in finding its cause, and the resistance which it offers to treatment. It has appealed to the popular imagination by the startling picture of its clinical manifestations. Much of value may be learned from a study

of the old writers. Lucretius[1] has supplied a graphic account in verse of a major convulsive seizure:—

> "Oft too some wretch before our startled sight,
> Struck as with lightning by some keen disease,
> Drops sudden: By the dread attack o'erpowered
> He foams, he groans, he trembles, and he faints;
> Now rigid, now convulsed, his labouring lungs
> Heave quick and quivers each exhausted limb.
> . . . . . .
> He raves, since soul and spirit are alike
> Disturbed throughout, and severed each from each
> As urged above distracted by the bane;
> But when at length the morbid cause declines,
> And the fermenting humours from the heart
> Flow back—with staggering foot first treads
> Led gradual on to intellect and strength."

We recognise in this picture the main features of the major seizure: the sudden onset, the fall, the cry, and the loss of consciousness; the tonic spasm, followed by the clonic convulsions, and the stertorous breathing. To these succeed attempts to move and to speak, a return to consciousness and the termination of the fit.

Hippocrates[2] has given a good account of the prognosis of the disease. He says:

"If it attacks little children, the greater number die. . . . If youths and young adults, recovery may take place, but there is danger of its becoming habitual, and even increasing if not treated by suitable remedies. Such also is the case when it attacks children. When it attacks people of advanced years it often proves fatal. When a person has passed the twentieth year of his life, the disease is not apt to seize him, unless it has become habitual from childhood. When the disease has prevailed for a length of time, it is no longer curable."

Galen[3] was the first to distinguish different varieties of epilepsy. He writes:

[1] Lucretius, "De rerum Natura," iii. fol. 88, Paris, 1514. Translated by Mason Good.

[2] Hippocrates, *Syd. Soc. Trans.* vol. ii. p. 850.

[3] Galen, "Ouvres Anatomiques, Physiologiques et Medicales," translated by d'Aramberg, tome ii. p. 571.

"In truth nearly all physicians have failed to distinguish between the different forms of epilepsy, of which there are three kinds. Disease of the brain is common to all epilepsies, whether the malady begins there, which is most common, or whether the sensations begin in the orifice of the stomach and ascend to the brain. There is another form of epilepsy which is more rare; this begins in any part of the body, and mounts to the head in such a manner that the patient is sensible of it himself."

I take it that we have here a differentiation between (1) the so-called idiopathic or genuine epilepsy, of which Galen distinguishes two sub-groups, the one more common in which the fit occurs without warning, the other with a gastric warning sensation, and (2) focal epilepsy characterised by localised convulsion and unassociated with loss of consciousness. So far as I am aware, this is the first reference to that form of convulsion which is permanently associated with the name of Hughlings Jackson.

Epilepsy began to excite a more widespread scientific interest, especially in France, in the closing years of the eighteenth and first half of the nineteenth centuries. We owe much of our knowledge of the disease to the writings of the French physicians of that productive period. Their names are well known: Tissot, Maisonneuve, Pinel, Esquirol, Morel, Moreau, Herpin, and Trousseau.

In the latter half of the last century, several writers in our own country gave much attention to the clinical study of the disease: Sieveking (1858), Russell Reynolds (1861), Hughlings Jackson (1863 and onwards), and Sir William Gowers (1881).

Within the last two decades, a renaissance in the study of epilepsy has taken place in all civilised countries, and a very large literature has sprung up around it in Germany, France, the United States, and Great Britain. It has recently attained the distinction of having a quarterly periodical devoted solely to its interests.[1]

[1] *Epilepsia*, Scheltema and Holkema, Amsterdam, 1909.

This renaissance has been largely aided by—(1) the study of the finer anatomy and the pathological changes in the central nervous system by the help of aniline dyes; (2) the great development in the study and knowledge of the metabolic changes within the body by means of chemical pathology; and (3) the greater facility of studying the life-history of epilepsy in all its manifold variations by the segregation of chronic epilepties in special institutions.

As I have elsewhere[1] given a detailed account of the clinical history and course of epilepsy, I propose in the present lectures to lay before you three particular aspects of the subject, namely:—

(1) The problem or nature of Epilepsy.
(2) The borderline of Epilepsy.
(3) The treatment of Epilepsy.

THE PROBLEM OF EPILEPSY.

The term epilepsy refers to a symptom; it is the clinical expression of a group of diseases which may be to a large extent differentiated from each other. They may be divided into four primary divisions:—

(1) The organic epilepsies.
(2) The early epilepsies.
(3) The late epilepsies.
(4) Idiopathic epilepsy.

(1) **The Organic Epilepsies.**—Under this heading are included—(*a*) Those forms of epilepsy arising from traumatic lesions of the skull, brain, or membranes. (*b*) Those which are associated with, or sequential to focal organic disease of the brain, such as tumour and thrombosis.

(2) **The Early Epilepsies** or the epilepsies of infancy and early childhood.[2]

[1] Aldren Turner, "Epilepsy," Macmillan & Co., London, 1907.
[2] H. Vogt, "Epilepsie im Kindesalter," Berlin, 1910.

These cases have been placed in a subdivision by themselves, although it is in many instances difficult to distinguish them from idiopathic epilepsy. Moreover, the two tend to fuse and overlap in later childhood. They are, however, tentatively taken out of the great group of genuine epilepsies, owing to the marked degree of mental impairment, amounting to imbecility or even idiocy, found in most cases. Many cases also are attributable to focal lesions—encephalitis, hæmorrhage, and thrombosis. A common variety is that form of epilepsy which is found with infantile cerebral hemiplegia. I have elsewhere shown that the greatest degree of dementia is found amongst those epilepties in whom the disease commenced before the fifth year of age, a group of epileptics also in whom structural stigmata of degeneration are most frequent.

(3) **The Late Epilepsies.**—Under this heading are included: —(*a*) The epilepsy associated with degenerative cardio-vascular disease. (*b*) The intoxication epilepsies, of which alcoholic epilepsy is the most common. (*c*) The epilepsy which occurs in the late stages of some dementing psychoses. (*d*) Epilepsy which springs out of eclamptic conditions, such as uræmia, puerperal eclampsia, etc. Puerperal eclampsia is, in many cases, the acute onset of epilepsy, which is continued in a chronic form by the recurrence of ordinary epileptic seizures, of the major or minor type.

(4) **Idiopathic Epilepsy.**—When these epilepsies have been eliminated, there still remains a large group, which has received the name of genuine, essential, or idiopathic epilepsy; a malady arising about the period of puberty, usually without any tangible or ascertainable cause.

Idiopathic Epilepsy.

Idiopathic or genuine epilepsy may be defined as a chronic disease of the brain, characterised by the recurrence of seizures in which interference with consciousness is an essential feature, associated either with convulsions or transient psychical phenomena, occurring usually in persons with a hereditary

neuropathic endowment, and eventually leading to more or less permanent mental deficiency.

This definition embraces the manifold symptoms usually included under the term epilepsy—such as transient jerks or jumps (*petit mal moteur*), aura sensations, incomplete attacks, so-called psychical epilepsy, automatism, the classical fit, and the psychical epileptic equivalents.

The sole feature necessary to establish the existence of epilepsy is sudden, brief loss or impairment of consciousness. Very characteristic of the epileptic attack is the temporary mental confusion, which succeeds the loss of consciousness. During this phase, semi-purposive actions or automatism may be observed.

In any discussion upon the problem, or nature of epilepsy, the two primary elements of the disease requiring consideration are—

(1) The Convulsive Element; and
(2) The Psychical Element.

1. **The Convulsive Element.**—The chief characteristic symptom of epilepsy is the periodic occurrence of attacks of loss, or impairment, of consciousness, sometimes accompanied by convulsions.

It has been laid down as a general principle, notably by Herpin in his classical, but little known work upon Minor Epilepsy,[1] that the symptoms occurring during the interval of the major seizures of this disease, such as cramps, "jumps," twitchings, spasms, partial convulsions, vertiginous attacks, cephalic sensations, visual hallucinations or other subjective phenomena, are the major or complete seizures reduced to their initiatory symptoms or sensations. All gradations of seizures may therefore be found in the same person, from the aura as the sole representative of the fit, through the incomplete attack to the fully developed epileptic fit, with the phase of tonic spasm, clonic convulsion, and the after-stage of coma. Let me illustrate my meaning by aid of a few simple examples—

[1] Herpin, "Les accés incomplets d'Epilepsie," Paris, 1857.

An epileptic described his attacks as beginning with a sensation in the epigastric region. The sensation was that of spasm or cramp in the stomach, and was sometimes associated with flatulence and nausea, and occasionally with a desire to go to stool. The spasm or sensation then took an ascending course towards the throat. When it reached the pharynx a sensation of choking was experienced, and the saliva collected in the mouth. About this period also an arrest of respiration occurred, probably from a tonic contraction of the respiratory muscles. A feeling of suffocation, usually associated with a sensation of fear and a desire to be alone then came on. If consciousness was not already abolished, the patient called out that he was going to have an attack, and was sometimes able to sit down. The sensation then passed rapidly into the head and consciousness was lost, but sometimes not before the convulsion had already commenced in the muscles of the head and neck. Then followed the usual phenomena of the major convulsive seizure (tonic and clonic convulsion, etc.).

In the intervals of such major seizures the patient experienced minor attacks, sometimes of the epigastric sensation alone, at other times of the epigastric warning with a further development towards the complete fit, namely, the choking sensation, the collection of saliva in the mouth, and the feeling of fear and cardiac oppression, but without the loss of consciousness.

I would especially call attention to these minor attacks, as they closely resemble attacks which will be described later on. Their epileptic nature is seen by the fact that they form the aura or initiatory symptoms of an epileptic fit, and by their rapid march—the upward passage of the sensation taking only a few seconds to reach the head.

All epileptics, whose major fits commence with a definite and constant warning—whether referred to the limbs, the head, the special senses, or of a psychical nature—have minor attacks consisting solely of the aura, or of the aura with a further development towards the complete attack. In other cases the warning is of a purely psychical character. One patient had as his minor fits, the aura of a "horrible sensation" which always

preceded the major attacks. In another the minor seizures consisted either of a "terrible feeling of a most disagreeable character," or of this sensation accompanied by obscuration of consciousness, and a fall, but without convulsion.

Attacks of this character are not uncommonly associated with what Hughlings Jackson has called "the dreamy state."[1] This may be of the nature of reminiscence; or it may consist of a sense of unreality, as if surroundings were unfamiliar, or of a sensation that what is happening has been previously experienced. The patient is frequently able to predict whether the incomplete or complete seizure is likely to occur from the intensity of the warning sensation. The feeling of intense fear with brief loss of consciousness, associated with its physical counterpart of running away as the means of escape, may form the whole of the attack.

In about two-thirds of all epileptics, there is no warning of the onset of the seizure, a sudden and complete loss of consciousness occurring either immediately before, or at the same time as, the onset of the tonic phase of the complete major fit.

The minor attacks occurring in the intervals of the complete fits in this group of epileptics are common—as there is no warning there is of course no incomplete minor seizure without interference with consciousness. The varieties of the minor seizures here form common types of epileptic fits.

A not unusual type of fit is temporary blurring of consciousness with muscular relaxation, so that the patient drops what he is holding, or conversation is momentarily suspended, or he falls. In other cases slight and partial convulsion may be observed. Some cases of this type present features of a purely psychical character, the movements noticed during the attack being not convulsive but automatic or semi-purposive.

The deduction drawn from a study of epileptic attacks, as just described, supports the conclusion of Herpin already given, that the minor attacks occurring in the intervals of the major fits are the initial symptoms of these attacks, which are arrested at a period more or less advanced.

[1] Hughlings Jackson, "Brain," 1898 and 1899.

From these few remarks upon the general outlines of epileptic attacks, especially of the minor type, it may be deduced (1) that some minor fits consist of the aura or warning alone, and are unattended by loss or even impairment of consciousness; (2) that others consist of the warning sensation with a development towards the more complete fit. These present a certain march of subjective symptoms, and terminate with, or without, some blurring or obscuration of consciousness; and (3) others present the complete or major type of fit.

If there is no warning, the minor attacks consist of lapses of memory, giddiness, falls, and "sensations" with or without slight spasm or convulsions, but always with some interference with consciousness.

Although the common clinical form of epilepsy is the combined type, or that in which minor or major seizures are found in the same person, yet other types or clinical groupings of fits are by no means unusual. Thus we find groupings limited to the major or the minor type of seizure respectively. There is also a type in which major or minor seizures occur in series of a score or more from time to time (serial epilepsy). Again, there is the acute form of epilepsy known as the *status epilepticus*, in which one fit follows another so closely that the after stage of coma gives place to the succeeding fit, without any return to consciousness.

From a study of these phenomena it would therefore seem logical to conceive the paroxysmal and interparoxysmal symptoms of epilepsy as links of a chain and not as isolated features. There may be found in the same person all stages of the fit, from the simple aura through the incomplete attack to the fully developed epileptic fit.

Whatever explanation therefore may be mooted as the immediate cause of a convulsive seizure must also explain the interparoxysmal spasms, cramps, and aura sensations.

Applying the same principle to the clinical groupings of fits, all degrees of combination may be studied from short series of incomplete minor fits, through serial outbursts to the picture of the acute status epilepticus.

Short series of minor or major fits are stages towards serial epilepsy which is merely a less acute and more drawn out form of status epilepticus.

2. **The Psychical Element.**—The recognition of the psychical element as a constituent feature of epilepsy has materially widened the range of epileptic manifestations. Some writers have gone so far as to relegate the convulsive element to a subsidiary position, and to regard "periodic fluctuations of the psychical equilibrium" as the outstanding feature of the disease. It is now generally accepted that certain paroxysmal psychical symptoms occurring in epilepties may replace from time to time the convulsive seizures, and form what are known as psychical epileptic equivalents; and that transient disturbances of consciousness of a psychical character may be the sole evidence of an epileptie attack (psychical epilepsy).

The psychical element in epilepsy may be studied under three conditions:—

(*a*) In the Epileptie Temperament.
(*b*) In the paroxysmal symptoms preceding, succeeding, or replacing the convulsions.
(*c*) In the Permanent Dementia.

(*a*) *The epileptie temperament* has many features common to epilepsy and the several forms of mental deficiency.

Epileptics are on the whole self-opinionated and egotistieal, possessing a conceit and assurance out of all proportion to their achievements. Their character is unstable, their mental perspective is disproportioned, they are sometimes conspicuous for their tenacity, which often reaches to obstinacy. Their conversation is usually prolix and pretentious. They frequently exhibit a religious fervour, which contrasts strangely with their actions, as their ideas of right and wrong are often vague. Like most mental defectives, their actions are characterised by indecision and doubt, by an exaggerated self-consciousness, by apprehensions and fears, by feebleness of will-power and hesitancy, which prevent resistance to certain impulsions and decisive aetion.

(*b*) *The paroxysmal psychieal phenomena* of epilepsy stand

in three ways to the convulsive attacks. Some are found as prodromal symptoms of the seizures, other as immediate sequelæ, and a third group, occurring in the interparoxysmal periods, are believed to replace the convulsions, and are in consequence known as psychical epileptic equivalents. The paroxysmal psychical symptoms are grouped under the general term of "epileptic insanity."

The pre-paroxysmal mental symptoms are in reality prolonged or modified phases of the prodromal stage. They consist of irritability, apprehensions, feelings of unreality, lethargy, and occasionally hallucinations. They may precede the fit by one or more days. On the other hand, they may consist of sudden impulsions, the patient doing some strange things and then suddenly falling down in a fit.

The post-paroxysmal mental symptoms are the immediate sequelæ of either single seizures, or more commonly of serial outbursts of the status epilepticus. They are inseparable from the convulsions, and disappear after a longer or shorter interval, but give in some instances a decidedly psychical character to the paroxysms.

The most frequently observed are—(1) Acute amentia or stupor, resolving slowly during the course of days or even weeks. (2) Acute mania, or excitement, of shorter duration than the preceding, but often of intense severity. Minor varieties of this are seen in phases of talkativeness, tiresomeness, and exhilaration of spirits. (3) Transitory delusional states; and (4) Automatism. Most minor fits are succeeded by some kind of automatic action, such as "exhibition," wandering or confusion of action.

The psychical equivalents are especially interesting forms of epileptic psychical phenomena.

The following features may be accepted as a satisfactory reason for regarding them as equivalents of epileptic convulsions:—

1. The occurrence in the family history of similar or allied conditions, interchangeable with attacks of genuine epilepsy.
2. Their association in the same person with the classical types of epilepsy, more especially at the catamenial epochs in women.

3. A certain uniformity in the type of the attack, recurring paroxysms reproducing the features of former attacks.

In any particular case the psychical equivalents usually correspond to the mental phases which are directly associated with the convulsions—(1) An epileptic, whose post-convulsive symptoms were irritability, quarrelsomeness, and pugnacity, had occasional transitory attacks of a similar kind without any antecedent convulsion. (2) Another had in the interparoxysmal periods attacks of the "dream state," which usually preceded his fits. (3) A third epileptic, whose post-convulsive symptoms were catatonic rigidity and stupor, showed from time to time in the intervals of his fits catatonic stupor states, unassociated with convulsion.

These are not uncommon types of psychical epileptic equivalents. But there are others; such are epileptic impulsions (*petit mal intellectuel*, of the French writers), acute manial outbursts (*grand mal intellectuel*), and transitory delusional states.

Psychical epilepsy.—Before leaving the subject of the psychical equivalents, I wish to say a few words about psychical epilepsy.

There is no doubt that a form of genuine epilepsy exists, characterised by short seizures of purely psychical type. These attacks consist of simple automatic actions performed by the patient in an unconscious state; the face is usually pale and has a fixed staring expression. There is no subsequent recollection of what has occurred during the seizure, although in some cases there would appear to be a state of sub-consciousness, in which the patient may hear vaguely what is said, but yet be unable to reply to questions. The attacks are characterised by sudden onset, and usually occur without warning. They are of short duration, and recovery is rapid.

They may be regarded as constituting one of the types of minor epilepsy, but differing from the common *petit mal* seizures by the purely psychical character of the fit. In the ordinary forms of minor epilepsy, the stage of automatic action is preceded by transient motor spasm or convulsion.

(*c*) *Epileptic dementia.*—In addition to the paroxysmal psychical symptoms, there is observed in all cases of confirmed or old-standing epilepsy, an almost constant and characteristic inter-

paroxysmal mental state, presenting features of great uniformity. The general features of this mental condition, or *epileptic dementia*, consist of all grades of mental deficiency, from a defect of memory, more especially for recent events, up to pronounced dementia. This should be regarded as an intregral part of the confirmed disease, modified to some extent by the duration, the frequency, and the character of the fits.

The existence of this dementia is of value in the differential diagnosis of epilepsy from some of the allied conditions which will be subsequently described (p. 36).

Course.—Epilepsy is most commonly found in a chronic form with periodic seizures recurring through many years, sometimes a lifetime, but in some cases showing prolonged and remarkable remissions. The frequency of the seizures may vary from one, two, or three in a year, to as many as a score or more in twenty-four hours.

A cure occurs in a small proportion of cases, estimated at from 10 to 12 per cent. These are cases in which the seizures are of infrequent occurrence, in which there is little or no mental impairment, and in which the bromide salts are well borne.

Having in this fashion briefly defined what is meant by idiopathic epilepsy, it is necessary now to ascertain whether there are any associated pathological changes in the brain or central nervous system.

Pathological Anatomy.

It is difficult to obtain post-mortem examinations on recent cases of the disease or on those cases characterised by seizures occurring only at long intervals; but it would appear as if genuine epilepsy was a disease which in its early stages was unassociated with any obvious structural changes in the brain or central nervous system. In old-standing cases, however, the morbid changes found after death are well recognised, and have been described by numerous writers. Dr. John Turner has found atrophy and sclerosis of some part of the central nervous system in considerably more than 50 per cent. of the cases of idiopathic

epilepsy. Alzheimer found acute and chronic changes in 60 per cent. of all cases examined.

In the *nerve cells* the changes vary from slight degrees of chromatolysis to advanced atrophic degeneration. The extent of the change would seem to depend largely, if not entirely, upon the mode of death. Certain nerve cells, showing well-marked eccentration of the nucleus, which is usually of a globose character, are stated by Dr. John Turner to be typical of low grade brains, and to represent an embryonic type of nerve cell. He regards these cells as stigmata of degeneration, and indications of a hereditarily acquired nervous instability.

The *vascular changes* consist of dilatation and atrophy of the blood-vessel walls, perivascular infiltrations, and punctiform hæmorrhages. The lymph spaces are dilated and contain a foam-like exudate. Hyaline spheres or lobulated bodies are seen lying free in the lumen of the smaller vessels and capillaries, and sometimes large hyaline masses form casts of the vessels in which they lie.

Neuroglia.—There is an increase of the glial tissue, especially that situated in the outer layers of the cortex, and of the cornu Ammonis. The meninges generally show slight fibroid thickening and a moderate leucocyte infiltration. Rupture of the vessels with extravasation of red blood corpuscles and a delicate unorganised foam-like exudate are always present.

The lesions found in the brains of epilepties are of wide distribution. The cerebellar membranes are often more affected than the cerebral. The glial changes are distributed unevenly over the whole surface of the cerebrum, but some regions are more especially involved, namely, the frontal and occipital regions, the cornu Ammonis, and the gyri around the fissure of Rolando.

It is now the generally accepted view that the changes just described are the effects and not the cause of the seizures. The acute or cell changes vary according to the manner of death, and the chronic or glial changes are dependent to a great degree upon the duration and severity of the disease. The most acute changes are found in the brains of persons dying in the status epilepticus (Alzheimer), and the most pronounced degrees of

gliosis are observed in the brains of those in whom the disease was of long standing and characterised by the severer grades of mental deficiency and dementia. Frequent repetition of epileptic fits brings about destructive changes in the nerve cells and fibres, whose place is taken by proliferated neuroglial elements. Hence there ensue, in the later stages of the disease, pronounced mental deterioration, and a slow and slouching gait, so well known to those conversant with the late stages of the confirmed malady.

In furtherance of the discussion upon the problem of epilepsy, three factors bearing upon its causation have to be taken into consideration. These are —

(1) The predisposing causes.
(2) The exciting causes.
(3) The immediate cause of individual seizures.

1. The Predisposing Causes of Epilepsy.

A statistical investigation of a large number of cases of epilepsy shows about an equal percentage without, as with, a history of ancestral neuropathic disorders. Even allowing about 20 per cent. for those in whom the family history may be either unknown, forgotten, or withheld, there still remains about 30 per cent. of all cases of epilepsy in which a history of family predisposition cannot be obtained.

Some explanation ought therefore to be forthcoming of so large a proportion of cases in which, on careful inquiry, no obvious heredity is obtainable.

The existence of epilepsy in some of these cases may be explained by the presence of a diathetic state, infective disorder, or intoxication in one or other parent at the period of conception.

It is not unlikely also that the presence of some symptoms, classified under the general term of "nervousness," may be of great importance in the genesis of epilepsy. Inquiries ought to be made into the question of parental neurasthenia, hysteria,

migraine or periodic headache, psychasthenia, obsessions, tics, chorea, and, in particular, those epileptoid symptoms which will be referred to in the account of the borderline of epilepsy. These are symptoms which do not, as a rule, strike the patient or his friends as of any importance in the causation of fits, nor does the physician usually direct his attention towards them.

A further explanation of some of these cases is to be found in the presence of small foci of encephalitis or of thrombosis occurring in infancy or early childhood. In many of these cases resolution has so largely taken place that the only permanent defect is some impairment in the finer movements of one or other hand. Defects of this character should always be looked for in those cases in which no clear predisposition is ascertained, or where the absence of well-marked "stigmata" suggests that a hereditary influence is not obtrusive. I find it impossible, however, to support the view of Freud, Marie, and others, that all cases of epilepsy are due to organic affections of a focal character occurring in early life, although it is quite clear that a small group of apparently genuine epilepsies arises in this way.

My earlier figures, taken from 676 cases of epilepsy, reveal the fact that the most common predisposing cause of epilepsy is ancestral epilepsy. In a further series of 214 cases, in which parental alcoholism, as a predisposing influence, was especially inquired into, similar conclusions were arrived at, namely, that ancestral alcoholism was a relatively minor factor compared with ancestral epilepsy in the causation of the disease.

TABLE I.—*Table showing the percentage of hereditary factors in the causation of Epilepsy.*

	First Series.	Second Series.
No heredity known . . .	330 or 49 per cent.	106 or 49·5 per cent.
Epilepsy	252 ,, 37·2 ,,	81 ,, 37·8 ,,
Insanity	37 ,, 5·4 ,,	7 ,, 3·2 ,,
Alcoholism	21 ,, 3·1 ,,	15 ,, 7 ,,
Other disorders	36 ,, 5·3 ,,	5 ,, 2·3 ,,
Totals . . .	676 or 100 per cent.	214 or 99 8 per cent.

This is a point of especial interest, when comparisons are made between figures obtained in different countries. The outstanding feature of Table II. is the enormous percentage frequency of alcoholism noted by the French observers, an ancestral disposition far outweighing the combined percentages of epilepsy and insanity, according to Dejerine's figures. The German figures, on the other hand, show the predominating influence of insanity, whilst the American give almost equal prominence to epilepsy and alcoholism.

TABLE II.—*Table showing the percentage frequency of Epilepsy, Insanity, and Alcoholism, as predisposing factors in Epilepsy.*

	Dejerine.	Binswanger.	Spratling.	Turner.
Epilepsy . .	21·2 per cent.	11 per cent.	16 per cent.	37 per cent.
Insanity . .	16·8 ,,	29·6 ,,	7 ,,	4·8 ,,
Alcoholism . .	51·5 ,,	22 ,,	14 ,,	4 ,,
No heredity . .	...	...	...	47 ,,
Totals . .	350 cases.	150 cases.	1070 cases.	890 cases.

STIGMATA OF DEGENERATION.

Evidence of a family neuropathic tendency may be found in what are known as "stigmata" of degeneration.

Structural stigmata have formed the subject-matter of considerable investigation since attention was directed to their importance by the writings of Morel and Moreau (of Tours). In the most pronounced forms of mental deficiency, such as are seen in imbeciles and idiots, anatomical variations from the normal are not uncommon.

In the slighter forms of neuroses, such as neurasthenia, migraine, and simple melancholia, the neuropathic stigmata are less frequent and less pronounced than are seen in the more exaggerated conditions above mentioned.

In epilepsy, as in the other degenerative neuroses, stigmata of degeneration are present, and have received considerable attention.

In addition to the cranial asymmetry which Laségue[1] considered to be constant in true epilepsy, Féré[2] has called attention to the frequency of cranio-facial asymmetry amongst epileptics.

Among other features of a degenerative character, the configuration of the hard palate deserves special attention. This has been the subject of much investigation and discussion, and its study and relation to the several degenerative neuroses is associated with the names, more especially in this country, of Langdon-Down and Clouston.[3]

Deformities of the ears have for long been regarded as important structural stigmata of degeneration.

Abnormal implantation of the teeth in connection with epilepsy and the developmental neuroses has recently been the subject of careful investigation by Talbot.[4]

In addition to the above-mentioned stigmata, Féré has also drawn attention to, and described in detail, various asymmetries of the thorax and pelvis, unequal length and size of the limbs and syndactylism, found in epileptics. In whatever form they are found, it has to be borne in mind that stigmata of degeneration are deviations from the normal, and occur in those who are subjects of a hereditary degenerative predisposition.

Of the two quinquennial periods in which the onset of epilepsy is more common, that from birth to five years of age is the most fruitful in stigmata. It is during this period that the growth and development of the brain is most active, and it is during this period, as Clouston has shown, that other degenerative neuroses and psychoses develop, such as stammering, backwardness in walking and talking, delayed dentition, and some forms of idiocy and imbecility.

Amongst epileptics, in whom the disease commences about the time of puberty, quite a considerable number present no obvious structural stigmata, even when parental epilepsy was

[1] Laségue, quoted by Féré, *op. cit.*
[2] Féré, "Les Epilepsies et les Epileptiques," Paris, 1890.
[3] Clouston, "The Neuroses of Development," Edinburgh, 1891.
[4] Talbot, "Irregularities of the Teeth," Philadelphia, 1901.

given as the probable cause of the disease. The most common stigma observed in epileptics is a deficiency in the physical development. Many epileptics are, on the other hand, physically well formed.

The general conclusions which may be made from a study of the stigmata of degeneration in epilepsy are—

1. That the absence of a family neuropathic history is of little account in face of well-marked structural signs of degeneration.

2. That the early epilepsies, or those commencing under five years of age, are associated with most marked mental defects, most obvious degenerative stigmata.

3. That stigmata are most common in that type of epilepsy known as the combined seizure type, which is the least tractable and most dementing form of epilepsy.

4. That stigmata are common in those cases of the confirmed disease which go on to marked dementia.

I therefore look upon the presence of well-marked degenerative stigmata in epilepsy as a factor of great importance. The future course of the malady may be gauged to some extent by their study.

Age at Onset.

The age at which epilepsy arises is an important factor in the study of the predisposing causes. Although there is no age-period which may be regarded as entirely exempt, there are certain age-epochs during which the onset of epilepsy is more common.

The first epoch is from birth to 5 or 6 years, or that period corresponding to infancy and early childhood. About 25 per cent. of all cases commence during this epoch. These cases are usually associated with marked mental deficiency or backwardness. Many of them are based upon organic lesions of the brain.

The second epoch is from 10 to 22 or 23 years of age, or that period corresponding to puberty and early adolescence. No less than 54 per cent. of all cases commence during this epoch. It

is *par excellence* the age of onset of genuine epilepsy. The mean age of maximum onset is 14 years, but the onset of the disease in young women is later by about two years than in the male sex.

The third epoch embraces the remainder of life. There is a rapidly lessening tendency towards the onset of genuine epilepsy from the age of 30 onwards. Most of the epilepsies occurring in this epoch are founded upon organic lesions, arterio-sclerosis, or intoxications (alcohol, lead, etc.).

From these facts it is obvious that genuine or idiopathic epilepsy is a disease of youth, coming on at a time when the development and growth of the central nervous system and the maturation of the organs of reproduction are taking place. It is during this period that causes, insignificant or insufficient in stable nervous systems, may light up the tendency to convulsion or epilepsy, in those hereditarily prone towards it.

2. The Exciting Causes of Epilepsy.

I have attempted to show that the predisposing cause of epilepsy is, in the majority of cases, an inherited neuropathic disposition, and that epilepsy may arise independently of any exciting or determining cause, being a spontaneous development attributable to the normal processes of brain growth and sexual maturation in predisposed persons.

In a number of cases, however, some exciting influence is at work in the production of the first epileptic seizure. It is a cardinal principle that the cause of epilepsy is that circumstance to which the first fit is apparently due. The instability of the brain once induced, becomes a characteristic feature, so that further attacks recur quite independently of the original or any other obvious exciting cause.

The common period for the onset of epileptic fits is puberty, and in girls its onset is frequently accompanied by irregularity in the catamenia. There is little or no evidence to support a popular view that the regular establishment of the monthly period is attended by arrest of the seizures in those in whom the

disease has arisen a few years earlier. The relation of epileptic fits to menstruation is well established. In most cases the seizures occur immediately before or shortly after the period; and in chronic cases the monthly period is characterised by an increase in their frequency.

The normal process of child-bearing in woman has also some influence in the causation of epilepsy. We find that *pregnancy* may be the exciting cause of the disease in some cases, that it may induce a relapse of the seizures in some cases in which a cure has apparently been established or in which a long remission has occurred. On the other hand, pregnancy has been known to produce a temporary arrest or amelioration of fits; and there are well-authenticated cases in which a permanent arrest has been brought about in this way.

Accouchement and the *puerperium* have also an important bearing upon epileptic fits. I have seen a number of cases in which the disease clearly originated at this time in the form of a puerperal eclampsia, and persisted for many years as confirmed epilepsy. These cases are interesting, as they argue in favour of the view originally propounded by Féré, that some types of puerperal eclampsia are merely the onset of epilepsy in an acute form in predisposed persons.

Sleep is another physiological condition eminently favourable to the onset of epileptic fits. In many cases fits only occur during sleep, so that a clinical type has been recognised as nocturnal epilepsy. There can be no doubt that cases of nocturnal epilepsy may exist unrecognised for many years, until a convulsion in the daytime establishes the existence of the disease. Voisin mentions the following phenomena as suggestive of the nocturnal form:—Intermittent wetting of the bed, occasional periodical and temporary mental dulness and irritability, well-marked lapses of memory, somnambulism, and unexplainable outbursts of temper. Pick has called attention to the frequency of fits during the hours of deepest sleep, namely, during the first hour or two after falling off to sleep, and again before waking. My own observations show that the greatest number of fits occur during the hour or two after falling off to sleep, a

period which probably coincides with the greatest degree of cerebral anæmia. Whether these facts are to be explained on the theories of brain anæmia (Pick), of lessened inhibitory control (Clark), of the alkaline tide (Haig), or of increase in the rate of blood coagulation,[1] are points on which evidence is insufficient.

A second and smaller group of epilepsies is found in which a definite extraneous cause acts as the excitant of the first fit, and, as such, of the disease. These causes may be—

1. *Psychical influences* such as emotional excitement, fear, shock, anxiety, grief, or overwork. Gowers, in particular, regards this influence as the most potent of all the immediate causes of epilepsy. It was the ascribed cause in only 4 per cent. of my cases, being twice as common in females as in males. In most of the cases a strong neuropathic predisposition was found.

2. *Infective diseases.*—The most frequent is scarlet fever, but measles, pertussis, influenza, and diphtheria have also been observed. An interesting feature in this connection is the occasional temporary arrest of the fits in consequence of an attack of pyrexia, probably due to the arrest in the chloride excretion with corresponding retention of bromide salts (Hoppe).

3. *Reflex causes* play an important part as excitants of epilepsy, more especially in children and young adults predisposed by heredity to nervous instability. I would especially call attention to the necessity of a careful examination of the nose (for adenoids, polypi, or foreign bodies), of the eyes (for errors of refraction), of the ears (for chronic otorrhœa), of the teeth, the genital organs, the stomach, and of the intestine (for the presence of worms).

3. The Immediate Causes of Epileptic Seizures.

From time to time it has been observed that the onset of an epileptic fit is accompanied by a temporary cessation of the heart's action and the pulse beat. This transient stoppage is

[1] Quoted by J. Turner, *Journ. Ment. Sc.*, London, 1908, October.

attributed to cardiac inhibition; the loss of consciousness and the fit to the resulting sudden cerebral anæmia.

The most recent advocate of the cardiac inhibition theory of the causation of epileptic seizures is Dr. A. E. Russell, who has collected a quantity of evidence in its favour in his Goulstonian Lectures for 1909.[1] He does not, however, contend that complete cardiac arrest occurs in every epileptic seizure, for the pulse may persist throughout a fit, although in it epidoses an extreme feebleness, short of absolute cessation, might be sufficient to initiate a fit.

It is well known that convulsions may be due to the sudden cutting off, or to the prolonged deprivation of the cerebral blood supply. The convulsions of sudden Adams-Stokes, the Kronecker-Tenner fits provoked by ligature of the internal carotid arteries, and the spasms consequent upon electrical stimulation of the peripheral end of the cut vagus nerve are all due to this cause. Russell maintains that the fundamental factor underlying the epileptic fit is cerebral anæmia, probably associated with vaso-constriction of the cerebral blood vessels. He holds that the sudden and profound loss of consciousness, so characteristic of the major epileptic seizure, is not due to an 'outrage' from the cortical grey matter, as is commonly stated, but to a sudden cessation of the whole cerebral circulation. This may vary in degree so as to cause either transient unconsciousness or a severe convulsive fit.

The direct evidence in support of this view is meagre, apart from the temporary cessation of the heart's action sometimes observed at the commencement of an epileptic fit. It would be therefore well to look for collateral evidence which might support the vaso-motor spasm theory of the origin of epileptic fits. A comparison has been drawn between what occurs in epilepsy and some of the phenomena of Raynaud's disease. The retinal arteries have been found on ophthalmoscopic examination to be narrowed during the paroxysmal attacks of impairment of vision which occur in some cases of Raynaud's disease. The fundus of the eye has also been examined ophthalmoscopically at

[1] A. E. Russell, *Lancet*, London, 1909, 1911.

the commencement of an epileptic seizure. Sir William Gowers did not observe any vaso-constriction of the retinal arteries under these circumstances, but some Italian observers have recently found such to be the case. Recurring attacks of transient hemiplegic paralysis have been described by Drs. W. Russell,[1] Langwill,[2] and others as due to intermittent closure of the cerebral arteries, a condition which might, if sufficiently sudden and complete, conceivably give rise to convulsions in predisposed persons. The well-known phenomena of migraine also are in many ways favourable to the hypothesis of vaso-motor spasm as the underlying cause.

Leonard Hill[3] produced a typical fit in himself by digital compression of one carotid artery. He says: "The first effect on applying the compression was a sensation in the eye on the same side; then there followed a sensory march of formication down the opposite side of the body. This began in the fingers, spread up the arm, and then down the leg. Finally, clonic spasms of the head occurred, accompanied by an intense feeling of vertigo and alarm."

The evidence in favour of cerebral anæmia being the cause of convulsions is quite clear, but as yet there is no direct demonstration of either vaso-constriction or cerebral anæmia being the exciting cause of the recurring seizures of epilepsy.

Paralysis of the vaso-constrictor nerves, rendered permanent by removal of the superior cervical ganglion, has not led to that favourable outcome which was expected of it when introduced as a means of treating epilepsy on the theory that the fits were due to cerebral anæmia from vaso-constriction.

Admitting the instability of the vaso-motor system as a feature of most epilepsies and vaso-constriction and cerebral anæmia as the cause of some fits, we have still to arrive at an explanation of the cause of a periodic tendency to arterial spasm.

Whether the primary excitant of the epileptic attack be a spontaneous "discharge" of nervous energy, or cerebral anæmia,

[1] Wm. Russell, *Practitioner*, 1906.
[2] W. Langwill, *Scott. Med. and Surg. Journ.*, 1906, June.
[3] L. Hill, "Cerebral Circulation," 1898, p. 124.

induced by vaso-constriction and cardiac inhibition, the fit would seem to originate in some portion of the cerebral cortex. I accept the old view of Hughlings Jackson, that epilepsy is a disorder of the highest level, and I contend that the observations of many subsequent workers, anatomical, experimental, and clinical, have placed the highest level between the intermediate precentral and prefrontal zones.

A. W. Campbell[1] has shown that in association with each recognised cortical centre there exists a corresponding higher or psychical centre. He has described the psycho-motor, psycho-sensory, psycho-visual, and psycho-auditory centre in close relation to the motor, sensory, visual, and auditory centres, situated respectively in the precentral, postcentral, occipital, and superior temporal gyri. If a higher olfactory centre exists, it is probably to be found in the lobus pyriformis, but its delimitation is less clear than in the case of the other centres.

The nature of the aura determines the portion of the cerebral cortex in which the fit starts. Warnings of special sense are either crude sensations or sensations of a more elaborate and psychical character. The latter are especially characteristic of idiopathic epilepsy. It is therefore conceivable that the "discharge" commences either in the lower or higher portion of a special sense region, according to the nature of the warning sensation.

A noteworthy point is the constancy of the warning in particular cases. As Herpin pointed out, the warning sensations, however diversified they may be, are always, or nearly always, similar in the same subject. There are a few exceptions. During the course of the disease, the aura of the major seizure may disappear, but the minor attacks retain the character of the original warning.

In the case of the large number of epileptics in whom no warning of the seizure is present, but in whom the fit is ushered in by a sudden and profound loss of consciousness, the discharge conceivably originates in the frontal area. Here the onset of the fit is essentially psychical. The patient is dazed, or

[1] A. W. Campbell, "Studies in Localisation," *Cambridge Univ. Press*, 1905.

stuporous, or drops down suddenly. In many of these cases, transient conjugate deviation of the eyes occurs, or a cry is uttered.

The completeness or incompleteness of an epileptic fit—that is to say, whether the attack is an aura sensation, a minor or a major seizure—depends upon the intensity, duration, and extent of the cortical discharge. Hughlings Jackson's idea of epilepsy was the occasional, sudden, excessive, violent discharge of a nerve centre, sensory, motor, or psychical. The conditions which favoured such discharge were congestion and excess of blood in the cortical capillaries, with retarded motion and consequent alteration of the nutrition of the cortical cells.

This view has lately been revived by the observations of Dr. John Turner, who has found in the brains of persons dying from epilepsy, minute thromboses of the cortical capillaries. He suggests that just prior to the onset of a fit the blood plates shed their nucleo-proteid contents. The result is the formation of granular or hyaline thrombi which, in persons hereditarily and structurally predisposed to cortical instability, is sufficient to provoke a cortical discharge and epileptic fit.

Whether in any particular case the morbid condition which gives rise to the "explosion" is inherent in a cortical centre, common to the whole cerebral cortex, or is attributable to a vaso-motor instability in blood condition, are points which still remain a mystery.

It may be asked: What is the cause of the enormous variability in the frequency of epileptic seizures, a frequency which varies from two or three fits in a year to several score in twenty-four hours? Is the same cause at work in both these types of the disease?

In whatever part of the cerebral cortex the discharge arises, it rapidly spreads so as to involve both sides of the brain, although one side is usually more affected than the other. Hence it comes about that examination of the reflex, motor, and sensory systems, after a major epileptic fit, reveals evidence of temporary paralysis, and of alterations in the reflexes, indicative of a transient disturbance of the pyramidal system. The knee-

jerks are at first abolished and then exaggerated, the abdominal reflexes are lost, and the plantars show a temporary extensor response. These symptoms vary in accordance with the character of the seizure. They are not present after minor attacks; they may be observed on one side only in cases where the convulsions have been preponderatingly unilateral. They are most obvious and persist longest after serial outbursts or the status epilepticus.

Temporary impairment of motor power (exhaustion paralysis) is observed on one or both sides, but more especially on the side which has shown the greater convulsion.

Diminution or loss of cutaneous sensibility may also be found after a fit. It is most marked in the limbs which show the greater motor weakness.

Amblyopia, deafness, and impairment of smell and taste may also be of temporary occurrence.

Toxæmic Epilepsy.

It has for a long time been contended that an excitant of epileptic seizures might be found in toxic or autotoxic causes, arising in connection with the body metabolism and gastro-intestinal disorders. The discrepancies, however, in the results of observations by different workers upon urinary and blood toxicity in epilepsy, have not afforded satisfactory evidence upon which to found such a theory.

It is now generally accepted that the altered condition of the urine and blood, which has been found in association with epileptic attacks by many observers, is the temporary effect and not the cause of the seizures.

Some explanation of these variations may be found in differences in the nature of the cases upon which the researches were carried out. Changes have not been found in the minor or *petit mal* type of the disease. Those cases which have shown the most pronounced reactions both of blood and urine, have been instances of serial epilepsy, the status epilepticus, and fits accompanied by acute mental symptoms—types of the disease in

which, from the general constitutional disturbances, there is more reason to suspect an infective or toxic cause.

Binswanger[1] has called attention to a small group of epilepsies which may have a toxæmic basis. This group is characterised by—(*a*) Well-marked premonitory signs, of which the most common are of a psychical type, irritability, fitfulness, quarrelsomeness, lethargy, somnolence, and delusions; (*b*) fits of frequent occurrence developing either into serial epilepsy, or the status epilepticus, accompanied by constitutional disturbances, such as furring of the tongue, constipation, acceleration of the pulse, and elevation of temperature; (*c*) more or less prolonged intervals free from attacks between the seizures.

I pass over with a mere reference the observations of Voisin and Peron,[2] who found a hypotoxic condition of the urine before and a hypertoxic condition after the fits; of Haig,[3] who laid stress upon the relation of epileptic fits to the normal diurnal variations in the excretion of uric acid; and of Krainsky,[4] who attributed the convulsions of epilepsy to the formation of carbamic acid, one of the derivates of urea. Details of the investigations of these observers have been given in my monograph on Epilepsy.

The presence of cholin in the blood and cerebro-spinal fluid of epileptics was originally described by Donath.[5] This substance, which was found by Mott and Halliburton[6] in the cerebro-spinal fluid in organic nervous disease, was attributed by them to disintegration of the myeline sheaths of the nerve fibres. Some observers hold that this is the cause of convulsions, acting as a toxic agent, as it has also been found in cases of general paralysis with epileptiform seizures. On the other hand, it has been looked upon as the result of the fits.

It has been shown experimentally (Dide, Donath) that moderate doses of cholin injected into the circulation do not

[1] Binswanger, "Die Epilepsie," Wien, 1899.
[2] Voisin and Peron, *Arch. de neurol.*, Paris, vol. xxiv. p. 178.
[3] Haig, "Uric Acid," 1892, p. 21.
[4] Krainsky, "Memoires Courronnés," 1901, p. 15.
[5] Donath, *Deutsche Ztschr. f. Nervenh.*, Bd. xxvii. part i.
[6] Mott and Halliburton, *Brit. Med. Journ.*, London, 1904, No. 2, p. 1557.

produce convulsions, but that in large doses both convulsion and paralysis may result. Buzzard and Allen [1] showed that the large doses of cholin necessary to produce convulsions in animals were much in excess of what could be produced under ordinary conditions in man. In a recent paper, Donath [2] records the results of his experiments into the convulsion-producing properties of the substances found in the blood and urine of epileptics. He showed that when injected into guinea-pigs and dogs, uric acid, neutral urate of sodium, carbonate of ammonium, kreatin, and lactic acid were entirely innocuous. The only convulsion-producing substances were ammonia and the organic ammonium bases—trimethylamin, cholin, kreatinin, and guanidin; the last body is not found in human urine, but is one of the uric acid products. A generally recognised feature of infection is increased coagulability of the blood. Dr. John Turner [3] has shown—(*a*) That the average rate of coagulation is quicker in severe cases of epilepsy; (*b*) that the rate is quickened during the period that the patient is having fits (serial epilepsy); (*c*) that there is a further quickening of the coagulation rate up to twenty-four hours before a seizure; and (*d*) that from twenty-four to forty-eight hours after a fit, there is a rebound and retardation in the rate of blood coagulation.

Intravascular clotting occurs clinically in cases of infection, most probably as a result of the liberation of nucleo-proteid. Dr. John Turner [4] has suggested that just before an attack the leucocytes and blood plates shed their nucleo-proteid. In consequence, a hyaline material is found in the smaller arterioles and capillaries, obstructing the free course of the circulation. These appearances are most conspicuous in the brains of persons dying in status epilepticus, as well as in general paralytics in whom epileptiform attacks have occurred.

It is therefore not improbable that some types of epileptic convulsions, notably serial epilepsy and the status epilepticus,

[1] Buzzard and Allen, *Rev. of Neurol.*, Edinburgh, vol. iii. p. 453.
[2] Donath, *Deutsche Ztschr. f. Nervenh.*, 1907, p. 232.
[3] John Turner, *Journ. Ment. Sc.*, London, 1908, October.
[4] John Turner, *Brit. Med. Journ.*, London, 1906, p. 496.

may be associated with nucleo-proteid thrombosis arising from toxæmic influences within the body. A further sign of toxæmia, described by Lewis Bruce [1] as occurring in some forms of acute insanity of toxic origin, is a hyper-leucocytosis, associated with a high percentage of polymorpho-nuclear cells. It would appear from the observations of Pugh [2] and Bruce that a hyper-leucocytosis was found in cases of serial epilepsy and those complicated with acute mental symptoms.

The general conclusion, therefore, which might be drawn from these observations is, that the type of epilepsy due to toxic causes may be found in those acute forms of the disease known as serial epilepsy and the status epilepticus. These may therefore be regarded as accidental occurrences in the course of the disease.

[1] L. Bruce, "Studies in Clinical Psychiatry," 1906.
[2] Pugh, *Brain*, 1902, p. 500.

LECTURE II.

THE BORDERLINE OF EPILEPSY.

In the sense used by Sir William Gowers[1] the term "Borderland of Epilepsy" includes some phenomena, which will not be discussed in this lecture, such as the minor seizures of labyrinthine disease, attacks often difficult to differentiate from epilepsy; migraine, which will not receive further consideration here, although the paroxysms of this disease are well worthy of study, as they reveal much that is interesting in the study of epilepsy; and vertigo, a general symptom of intracranial disease, which would lead us too far away from the prime object of these lectures.

I propose, therefore, to limit my remarks to those somewhat rare phenomena, which may truly be said to occupy the borderline of epilepsy.

Epilepsy and Hysteria.

In any discussion, therefore, of the phenomena on the borderline of epilepsy, brief consideration should, in the first place, be given to hysterical seizures and their differentiation from epileptic attacks.

Both epilepsy and hysteria are diseases founded upon a hereditary degenerative endowment. Both disorders have a common clinical symptom, namely, the periodic occurrence of paroxysmal attacks, characterised by an alteration of consciousness. This is hardly the place to enter into an analytical consideration of the distinction between the two great psychoneuroses, epilepsy and hysteria, apart from their paroxysmal phenomena.

[1] Sir William Gowers, "The Borderland of Epilepsy," London, 1907.

I have in the preceding lecture attempted to point out what may be regarded as the outstanding symptoms of epilepsy, and have endeavoured to show that although in recent and slight cases it may remain throughout a so-called "functional" disease, in the severe and the confirmed cases with mental impairment, the symptoms become grafted upon an organic alteration of the cortical tissues. Epilepsy, therefore, is a dementing disorder. Hysteria, on the other hand, remains throughout a purely functional, mainly psychical, disease. According to Janet, its fundamental characteristic is a dissociation or severance of some mental processes from the main personal consciousness. It has no pathological anatomy, and no tendency towards dementia.

Increasing clinical study of the attacks which occur in these two diseases has led to difficulty and confusion rather than to simplification in their differential diagnosis. The most recent observations tend to show that there is no such generally recognised condition as "hystero-epilepsy" in the usually accepted sense. Binswanger states that interparoxysmal hysterical symptoms may be added to genuine epilepsy, and *vice versa*. It would also appear probable that a prolonged hysterical attack may terminate with symptoms in no way differing from those of genuine epilepsy.

Bratz and Falkenberg [1] found that all their cases were either hysteria or epilepsy, or a combination of the two diseases. Major hysterical attacks are, as a rule, fairly distinct in character from major epileptic seizures. But the minor attacks of both disorders are sometimes not easy to differentiate. If an attack is being described by an untrained onlooker, it may be a difficult matter for a physician to decide whether the paroxysms are those of epilepsy or hysteria, as the duration of the minor seizures in both diseases is almost the same, and they may both occur in series. Moreover, the perversion or dissociation of consciousness in the hysterical attack may readily be mistaken for the abolition or impairment of consciousness which is the characteristic feature of the epileptic seizure.

[1] Bratz and Falkenberg, *Arch. f. Psychiat.*, Berlin, 1903, S. 328.

On the other hand, if the attack should be observed by the physician, there is, as a rule, less difficulty. The rolling or squinting movements of the eyeballs, and the oscillations of the head in the hysterical attack, are quite distinct from the tonic conjugate deviation of the head and eyes in the epileptic; the tonic spasm of the limbs inducing flexion of the arms and extension of the legs in the latter, are in contrast to the tonic spasm mainly of the muscles of the neck and back in the former; the steady development and eventual yielding of the clonic spasms in epilepsy stand out in striking contrast to the vibratory tremors of the hysterical attack; and the sudden relaxation of the spasm and the almost instantaneous return to consciousness in the hysterical seizure is a feature of noteworthy importance. The hysterical seizure differs essentially from the epileptic in the three following additional features:—(1) The hysterical seizure does not terminate in psychical disturbance, confusion, or automatism. Hystericals come out of their attacks suddenly, and often express a feeling of relief after the attack is over. (2) As pointed out by Janet, there is a possibility of awakening the phenomena of the hysterical seizure by hypnotism. Epileptics are not at all hypnotisable, and in consequence it is not possible to produce in them a real somnambulistic state with consecutive amnesia. (3) Should it be possible to examine the reflexes of the patient recovering from a seizure, certain changes will be found in most cases of major epilepsy.

The most important transient changes indicating disturbances of the pyramidal system are alteration of the plantar reflex from the normal flexor to the abnormal extensor response, abolition or impairment of the abdominal reflexes and exaggeration of the knee-jerks.

If, therefore, it is not always possible to distinguish the epileptic from the hysterical seizure, during the attack, an examination of the patient at or immediately subsequent to its termination will usually be sufficient.

Epileptoid Phenomena.

I now enter upon a subject of great complexity. There is no doubt that many people are prone to attacks which cannot be classified either under hysteria or epilepsy. The attacks are in the main purely subjective, but in some cases, objective phenomena, chiefly of a circulatory and vaso-motor character, may be observed.

Numerous writers, whose special work lies chiefly in neurological practice, have described attacks having features which will be depicted presently. Owing to the absence of any systematic classification, there has been a tendency to group the symptoms under different headings, each author devising his own terminology, for the sake of convenience or according to his conception of the malady.

Oppenheim was the first to describe certain periodic seizures under the term "psychasthenic attacks," mainly because they occurred in persons subject to common psychasthenic symptoms, such as fears, dreads, apprehensions, obsessions, and tics.

The leading features of these attacks were—(*a*) That they did not occur during childhood; (*b*) that there was a special cause for the attacks; (*c*) that they were of infrequent occurrence; (*d*) that they were characterised by prolonged unconsciousness without convulsion, and only slight twitching of a few muscles; (*e*) that there was an absence of mental changes.

These attacks would seem to be extremely rare, and in my opinion difficult to differentiate from epilepsy.

Janet[1] has described under the term "psycholepsy" conditions characterised by an alteration of consciousness, which favours the development of a "dreamy state," in which a feeling of unreality or of non-existence is the outstanding symptom.

Mention has already been made of the "dreamy states" described by Hughlings Jackson as the warning of some fits, with epigastric and olfactory auras. It is important to distinguish between this aura sensation and the condition which is now

[1] P. Janet, "Les Obsessions et la Psychasthenie," Paris, vol. i.

being described. Patients describe the mental state as a feeling of non-existence, with doubts as to the reality of things, even as to the material existence of their own bodies; they also have doubts as to whether what they see and hear are real. It may more appropriately be termed a "reverie" state, and is frequently associated with feelings of an obsessional or psychasthenic character.

Symptoms of this kind are not uncommon iu epileptics. Crichton Browne and Pick (of Prague) have described them as becoming permanent in some cases of confirmed epilepsy, and I have seen them as psychical epileptic equivalents.

Sir William Gowers,[1] in his most instructive lectures upon the "Borderland of Epilepsy," has described seizures whose symptoms were referred chiefly to the distribution of the pneumogastric nerve. To these he has given the term vagal or vaso-vagal attacks.

Finally, Dana [2] has designated under the term "para-epilepsy" seizures of a psychical, vertiginous, or vaso-motor type, occurring in neurotic or psychasthenic individuals, more especially about adolescence. As these seizures, according to this observer, resemble "aura fits," or abortive epileptic attacks, he has classed them in a group of para-epileptic phenomena.

It is therefore clear that observers now recognise a type of periodic seizure, which is neither hysterical nor epileptic, occurring in persons of a neurasthenic or psychasthenic temperament. Attacks of a not dissimilar kind, however, may be observed in subjects of genuine epilepsy, and there are other attacks in which the diagnosis from hysteria is most difficult and uncertain.

These attacks may therefore be provisionally placed in a group by themselves, but it seems to me as if a name is still lacking which will fitly embrace the whole series.

I have suggested the term "epileptoid" as one which seems to include all the symptoms of a paroxysmal and recurring character, which lie upon the borderline of epilepsy.

[1] Sir William Gowers, "The Borderland of Epilepsy," London, 1907.
[2] Dana, "Epilepsy and Epileptics," U.S.A., 1908, p. 90.

It would seem possible from our present knowledge to subdivide these symptoms into two groups—(*a*) Attacks having features of a vaso-motor character; (*b*) attacks having symptoms of a psychical character.

1. **Vaso-Motor Attacks.**—These seizures are characterised by throbbings, thumpings, and beatings of the heart, flushings of the face, and a fulness of the head, dizziness, and, frequently, sensations of a paræsthetic character in the extremities.

They may be of daily occurrence, and last from a few minutes to several hours. They occur in persons of a nervous temperament. Dana notes that there is usually an associated element of thyroidism. In one case of my own there was a decided tendency towards dermographism.

Gowers also describes seizures characterised by definitely vaso-motor symptoms, such as coldness of the limbs, pallor of the face, shivering almost amounting to rigor, tingling and numbness of the extremities, and sometimes a slight tetanoid spasm.

A fuller development of these seizures bringing them into a relationship more akin to epilepsy is well illustrated by the vaso-vagal attacks described by Sir William Gowers.

The symptoms of the vagal attack as given by Gowers are—(1) Sensations referred to the stomach, heart, and respiratory systems. (2) The ascent of the sensation from the stomach to the chest, throat, and head. (3) It is accompanied by a feeling of respiratory distress and cardiac oppression, fear, and a sense of impending death. (4) There is no true loss of consciousness, but the mental operations are slow, and sometimes characterised by a feeling of unreality. (5) The attack ends with a great acceleration of the heart's action; and (6) The whole attack lasts for about fifteen or twenty minutes.

"Women suffer more frequently from these attacks than men. This, and the fact that the pneumogastric and vaso-motor systems are readily influenced by emotion, have probably led to the frequent submergence of these attacks beneath the vague conception of hysteria!" (Gowers.)

2. **Psychical Attacks.**—These are seizures in which the outstanding features are of a psychical character, usually a sense

of apprehension, dread, or fear. They are commonly associated with sensations of nervousness, numbness, coldness, nausea, cephalic sensations, and frequently accompanied by sensations of a vague indescribable character. Sometimes the feeling of fear is intense, and may be replaced by a sense of impending death.

In the true attacks of this kind consciousness is never abolished, but the mental state may assume a dreamy attitude, with a feeling of unreality.

The attacks occur in persons who present other features of a psychasthenic character, such as dreads, fears, and apprehensions. There is usually also a well-marked neuropathic heredity.

Although in many cases the attacks are of the kind just described, there are other cases in which these attacks merge into epileptic seizures.

The attacks described here as "psychical" are clearly closely related to those of a more purely vaso-motor type; the vaso-vagal attacks of Gowers being seemingly the connecting link between the two types of seizure, as well as between them and attacks of an epileptic nature.

This is a suitable place to draw attention to some of the outstanding features of these attacks or to compare them with epilepsy.

One of the commonest forms of warning in epileptic seizures is the epigastric sensation consisting of a subjective sensation referred to the stomach or upper abdominal area. Epigastric sensations are of various kinds. Some are vague and indefinite, others are of an indescribable character, and others are painful, spasmodic, or cramp-like. These sensations may or may not be associated with gastric disturbances, such as flatulence or any abnormal condition of the stomach contents. In the majority there are no signs of local gastric disorder.

Closely allied to the epigastric sensation is the cardiac warning, which consists mainly of palpitation of the heart, and sometimes of pain, and is generally accompanied by a sensation of suffocation, strangulation, or oppression, sometimes with a sensation of impending death. So closely does the condition

resemble angina pectoris, that a variety of minor epileptic attacks has been designated "epileptic pseudo-angina."

A notable accompaniment of the epigastric warning and its allied sensation of cardiac oppression is sensation of intense fear or apprehension, and sometimes a desire to be alone.

The symptom of fear, in association with fits accompanied by epigastric warning, is well known, and was pointed out by Hughlings Jackson many years ago. But it is not confined to fits with epigastric warning. It is also found as a very characteristic feature of that remarkable group of fits known as "uncinate seizures"; or epileptic attacks with a warning of smell and taste, due to organic disease of the uncinate gyrus.

In addition to the sense of fear, which has been already mentioned, a peculiar "dreamy state" has been described, in which consciousness is lost or perverted.

Whatever may be the explanation, it would appear to be a well-recognised fact that a definite relation exists between epigastric sensations and a sensation of fear or apprehension. This relation is not confined to epileptic attacks. It is also present in other conditions. In many states of fear or alarm a vague sensation is referred to the abdomen. Persons who suffer from depression constantly refer to the feeling of weight or oppression in the region of the stomach; and many individuals, obsessed by some fear or panic, refer to a vague and indefinite sensation in the stomach as the source of their discomfort.

If we now apply the principles which have been already laid down when considering the march of an epileptic attack, it is obvious that minor epileptic seizures may occur, characterised by an epigastric or cardiac sensation, accompanied by a sensation of fear, or of oppression or even of impending death, in which consciousness may be retained, or at the most merely blurred or perverted.

I have ventured at some length into this discussion, because, as Dana has shown, the attacks already described as "psychical seizures" have many of the qualities of epileptic auras and abortive epileptic fits; hence the term "para-epilepsy" which he has applied to them.

The close relation between epilepsy and the vaso-vagal attacks has also been emphasised by Sir William Gowers, who regards them as a long-drawn-out or extended epileptic seizure.

He says: "Some of the facts we have glanced at suggest that it is possible that the elements of an epileptic attack may sometimes be extended, and thereby made less intense, though not less distressing. . . .

"If we could conceive a minor epileptic attack that is extended, its elements protracted with no tendency to be terminated by loss of consciousness, its features would be so different that its nature would not be suspected. Swiftness is an essential element of ordinary epilepsy, but this does not preclude the possibility of deliberation."

Dr. A. E. Russell, who has already been referred to as the most recent supporter of the cardio-inhibitory theory of epileptic seizures, interprets those attacks in the following way: "A sudden depression of the normal vaso-motor tone in the splanchnic area leads to a rapid accumulation of blood in the capacious vessels of that region. If only momentary in duration, the result is merely the sudden sinking at the pit of the stomach. If of longer duration, this preliminary sensation or aura is succeeded by a faint. . . . The dreamy mental state and the fear of impending death may be due to the vascular depression, which must of necessity affect the brain. If consciousness is lost, it shows that the vascular depression has reached a point incompatible with consciousness, and that this is the missing link connecting it with epilepsy."

The position of these seizures amongst periodic phenomena therefore depends upon the view which is taken of the nature of epileptic attacks. Russell regards the whole series from simple syncopal attacks or faints up to epilepsy as primarily of vaso-motor origin. He favours the doctrine that such symptoms are signs of "a vaso-motor ataxia," a condition in which the vascular mechanism is unable to adjust itself to strain.

Whether this is the true explanation of a remarkable series of phenomena is a point upon which it is difficult to offer an opinion. The theory just expressed certainly brings a train of

not dissimilar periodic seizures into line. Epilepsy, however, seems to me to be so essentially a cerebral or psychical disorder, that I hesitate to classify it along with faints and syncope as a disease of the vaso-motor mechanism.

Many of the features of the borderline conditions just described are not psychical, but seem to be the consequences of functional disturbance of the lower centres in the medulla oblongata. I would also emphasise the fact, already referred to in the first lecture, that the convulsive element is only one feature of epilepsy. True epilepsy is always accompanied by some psychical alteration, apart from symptoms of a psychasthenic kind. I am not aware that the epileptoid conditions just described ever present any tendency to mental impairment or dementia.

Sleep Symptoms.

The physiological condition underlying sleep is especially favourable for the development of epileptic attacks. It was shown in the first lecture that the few hours after falling off to sleep are the hours most favourable for the occurrence of epileptic fits. In consequence of this there is in many cases of epilepsy a striking regularity in the periodicity of the attacks, a fit occurring at or about the same hour on each occasion. This period of deepest sleep is also a favourable time for certain sleep symptoms which appear to be upon the borderline of epilepsy, namely, *night terrors in the adult.* Night terrors are common in neurotic children, but their significance is merely that of an unstable nervous system. Their occurrence in adults, or their continuance from childhood into adult life, is of greater significance, and may raise the question of their epileptic character in some cases. They usually reveal themselves in the utterance of loud piercing screams, sometimes a single scream, at other times more frequently repeated. Their chief characteristic is that having occurred at some time during the first few hours after falling off to sleep, they do not occur again during the same night. They have been known to recur every night for many months in succession; on the other hand, they may recur only at long

intervals. In most cases the sufferer is entirely unaware of their occurrence, except in those cases in which sleep-walking also occurs, where the patient awakes and finds that something unusual has taken place.

I have known cases in which the attack has been associated with a dream, but these are more likely to be cases of epilepsy.

Sleep-talking and sleep-walking either replace or may be associated with nocturnal screaming. Adult patients subject to these and similar sleep symptoms, sometimes present the stigmata, both structural and psychical, which are characteristic of epilepsy. In all, there is a well-marked neuropathic heredity. It is well known, however, that attacks in which a patient may wake suddenly from sleep in a state of nervous fear, apprehension, or panic, are of temporary occurrence, and indicate a state of nervous stress or strain, induced by overwork or anxiety, and not infrequently excited by some gastric or intestinal disorder.

I wish, however, to point out that sleep symptoms are found, from time to time, in persons of highly nervous temperament, which should be regarded as symptoms of importance and probably classed as phenomena upon the borderline of epilepsy.

LECTURE III.

TREATMENT OF EPILEPSY.

The treatment of so multiform and persistent a malady as epilepsy requires to be considered from several points of view. What might be called the hygienic and disciplinary treatment was more seriously carried out in the days before the introduction of the bromide salts than at the present time.

Celsus,[1] who gives a very meagre account of the disease, has some suitable remarks to make upon the general handling of a case of epilepsy. He gives some useful hints upon diet and general hygiene, with special reference to exercise. He says:

"The diet ought to be of the middle kind, for the case requires strength, and crudities are to be guarded against. He should avoid undue exposure, wine, venery, and all business. The body may be gently rubbed in the morning. Then let him take a walk as long and as straight as possible; after the walk let him be rubbed in a tepid place, not less than two hundred times, let him eat a little, and then rest."

The French physicians of the early part of the nineteenth century used a large assortment of drugs, and, on the whole, obtained good results, but they also submitted their patients to dietetic formulæ and general discipline.

Let me quote the method of treatment prescribed by an English physician during the years immediately preceding the introduction of the bromides in the treatment of epilepsy. Sir Edward Sieveking,[2] in his work on Epilepsy, published in 1858, writes:

[1] Celsus, "On Medicine," Dr. Greive's translation, Book III. Chap. xxiii. p. 168.
[2] Sieveking, "On Epilepsy," London, 1858, p. 229.

"If I were to formularise the prevailing mode of treatment which I myself adopt, I should say it consisted in local derivation and counter-irritation directed against cerebral congestion, and in general roborants or tonics. The selection of the special mode in which the latter indication has to be carried out necessarily depends upon the results of the inquiries into the condition of the individual organs.

"The great prevalence in epilepsy of headache either as an habitual affection, as a precursor to the attack, or as a sequel accompanied by other symptoms indicating a congestion of the head, suggests attacking the head by more or less severe counter-irritants. . . . I am also satisfied that in some of our cases the apparent cure was mainly due to the application of setons.

"The air the patient breathes, the water he drinks, and his ablutions, his daily occupation and habits, his amusements, his food and beverage, his clothing, his mental and moral history, and his prospects in life, should be inquired into in order to determine, whether or in how far, one or more of those elements require modification."

Since the introduction of the bromides by Sir Charles Locock[1] in 1857, most epileptics at some period in the course of their malady have probably been treated by these drugs. The universal prescribing of the bromides during the past half-century with more or less success in subduing the convulsions, has to a large extent deprived the patients of the advantages which may be obtained from the hygienic, dietetic, and disciplinary lines of treatment.

As the physical and mental development of epileptics varies as much as in conditions of health, it is obvious that lines of treatment suitable for the physically robust and mentally capable may be detrimental to the physically frail, and impossible in the dementia of the confirmed disease. We have therefore in every case of epilepsy to treat the individual and not solely the disease.

1. It is well known that epilepsy may undergo spontaneous cure even after the convulsions have lasted for a number of years.

[1] Locock, *Med.-Chir. Trans.*, London, 1857.

2. There is a considerable precentage of epilepties in whom the disease is eharaeterised by the infrequent oeeurrenee of fits, and the absenee of any obvious mental impairment. These eases eonstitute a favourable type of epilepsy, and may be treated medieinally and dietetieally on the lines whieh will presently be laid down.

3. In a large number of eases the disease would seem to be exeited in predisposed persons by reflex irritation of the peripheral organs. I would espeeially eall attention to the value of a eareful examination of—(*a*) The nose, for foreign bodies or adenoid growths in ehildren, and nasal polypi in adults. (*b*) Of the eyes, for errors of refraetion (whieh are partieularly frequent in epilepties), and their eorreetion by suitable glasses. (*c*) Caries of the teeth requires the necessary attention, as I have frequently observed great improvement in the fits after the adjustment of suitable artifieial means of mastieation. (*d*) All disorders of the stomaeh and intestines: dyspepsia, constipation, and worms should be earefully attended to. Epilepties being notoriously big eaters, require great eare in the regulation of their meals, and should be eneouraged to fully mastieate their food. (*e*) Loeal disease of the pelvie organs in women requires attention, but too much stress ought not to be plaeed upon those disorders even in predisposed persons, although their treatment by suitable remedies should be earried out.

4. There are many eases of epilepsy in the early stages of whieh treatment in an institution may be either impossible or unadvisable. Under those eireumstances the patient ought to be plaeed under the eare of a eapable and properly trained nurse-attendant. The satisfaetory earrying out of this instruetion will often eventuate in most gratifying results.

5. Epilepties suffer notoriously from lowered vitality and sluggish eireulation in the extremities. For those, warm baths, spinal douehes, and massage are desirable. The use of the hot bath is most neeessary not only for the ordinary ablutionary purposes, but to promote skin exeretion, which is regarded by some writers as being of a toxie nature. Under all eireumstanees a eertain amount of daily exereise in the open air is neeessary;

walking, running, tennis, croquet, and football, may all be indulged in freely. It is scarcely necessary to point out that such exercises as bicycling, rowing, swimming, and riding are to be avoided.

6. An out-of-door life is usually regarded as the most suitable for an epileptic, but this entirely depends upon the physical vigour of the patient; hence farming or market-gardening should only be recommended to those of a robust constitution, while for the frail epileptic, drawing, modelling, book-keeping, and such semi-sedentary forms of work are quite suitable. On the other hand, there are many who are able to continue their professional work or business without difficulty, and with advantage to themselves and with benefit to their disease.

7. I do not think it right that those subject to even infrequent epileptic seizures should be sent to school along with healthy boys and girls. In all cases private education is to be recommended. In cases with marked mental impairment, special methods of teaching ought to be adopted.

8. If the disease develops during a period of mental strain preceding an examination, complete abstinence of work should be temporarily enforced, but when the type of the disease has revealed itself, education should be resumed on the lines already laid down.

9. The marriage of epileptics should be discouraged. There is a popular belief that if an epileptic girl is married the disease will thereby be cured. I have met with isolated instances of this, but in the majority of cases pregnancy and the puerperium are especially prone to increase fits, or at the best to exert no change at all upon them.

It is now necessary to enter with some detail into the medicinal and dietetic remedies commonly adopted in the treatment of epilepsy.

In the first place, I wish to emphasise the importance of separately considering the treatment of recent epilepsy from that of the confirmed disease.

Let me therefore first discuss the treatment of epilepsy at the onset of the disease, or in its early stages.

The Bromides.

There is no single specific remedy in the treatment of epilepsy, although the alkaline salts of bromine come nearest to this definition. But the influence of the bromides upon epileptic convulsions is variable and uncertain.

In the first place, *bromide medication may arrest the seizures immediately*, or within a short period of their administration, temporarily or permanently. In this division most of the curable types of epilepsy are found: cases characterised by an absence of mental impairment and with fits recurring only at long intervals. If any given case is capable of arrest, a satisfactory response will be apparent within a comparatively short period of commencement of the bromide treatment. In eighty-six cases, in which the fits were arrested for periods varying from two and a half to twenty-two years, rather more than 50 per cent. yielded to treatment within the first twelve months of regular bromide administration. (Out of 366 cases of epilepsy, eighty-six, or 25·5 per cent., belonged to this class.)

Secondly, the bromides may induce a *lessening in the severity and frequency of the seizures.* This is the common temporary result of bromide treatment, and is what may be confidently expected in the majority of cases in the early stages of the disease. Sometimes the change is effected by the arrest of the major seizures, the minor continuing; or the bromide may change the time-incidence of fits from the waking to the sleeping hours, or *vice versa.* (In the series of 366 cases of epilepsy in which bromide medication was continued over long periods, 105, or 28·7 per cent., belonged to this class.)

Thirdly, *the bromides may exert no influence at all upon the disease,* or may even augment the frequency, or severity, of the seizures. (In the series already quoted, 175, or 47·8 per cent., remained unimproved and became confirmed epilepties.)

These numerical facts may be given in the form of a table

showing the general results of treatment by the bromide salts in 366 cases of epilepsy.

TABLE III.

Cases of arrest	86	or 23·5	per cent.
„ showing improvement .	105	„ 28·7	„
Confirmed cases	175	„ 47·8	„
Total .	366	„ 100	„

These statisties are in general harmony with the observations of other writers on the subject; they demonstrate elearly how large a percentage of epilepties derive no benefit from bromide treatment. This is within the knowledge and personal experienee of most physieians, who have had to treat even small numbers of epileptic patients. A further eonclusion drawn from the study of these figures is, that under the influence of the salts of bromide a eonsiderable number of cases of epilepsy are temporarily "eured," or materially improved. It is, however, within the experienee of most observers that no eomplete reeord ean be obtained of the total number of eases which are relieved by these means, as, more especially amongst hospital patients, there is a tendency, onee the fits have been temporarily arrested, for the patient to eease attendanee. Notwithstanding the unfavourable results of treatment in many cases, it would be an error to say that bromides are useless in the treatment of epilepsy. If 50 per eent. of the eases derive benefit from the administration of these drugs, then all eases, especially of reeent onset, should be given the benefit of the drug for a time. It is not the bromide salts as a remedy, but the method of their administration, whieh is harmful, and to this attention will be directed immediately.

Bromism.—The physiological action of the bromide salts eonsists in lessening the irritability of the eentral nervous system, and in exerting a subduing effeet upon reflex aetivity and cerebral function. The potash salts of bromine also induce a slowing of the pulse and the action of the heart. In medium

doses (30 to 60 grs.) the bromides produce muscular fatigue, a slowing of the mental processes, dulling of the sexual function and of the skin sensibility. In large doses (150 to 225 grs.) the speech becomes slurred, there is abolition of the palatal and pharyngeal reflexes, while frontal headache, and a limitation of the power of thought, soon ensue. Salivation, lowering of the body temperature and of the pulse rate, catarrh of the stomach and of the respiratory mucous membranes, are general bodily symptoms resulting from continual use of large doses of the bromides. It is therefore obvious that the prolonged and injudicious use of the bromides may give rise to the toxic symptoms known as *bromism*. This condition is characterised by a blunting of the intellectual faculties, impairment of the memory, and the production of a dull and apathetic state. The speech is slow, the tongue tremulous, the saliva may flow from the mouth, the gait is staggering, and the movements of the limbs feeble and infirm. The mucous membranes suffer so that the palatal sensibility may be abolished, and nausea, flatulence, and diarrhœa supervene. The action of the heart is slow and feeble, the respiration shallow and imperfect, and the extremities blue and cold. An eruption of acne frequently covers the skin of the face and back.

Dosage.—Bromide treatment should be commenced at the earliest possible time after the onset of the fits, as there is a greater prospect of arrest or improvement during the first five than during the second five years of the disease, although arrest of the seizures may occur after a duration of twenty years. The administration of the bromides should be continued for a period, the duration of which is to be determined by the study of each case separately, but should not be less than two years. The dose usually given is, I think, too large. If benefit does not follow a daily dose of from 45 to 75 grs. of one, or a combination of the bromide salts, some other remedy or method of treatment should be sought for and applied. Dr. Clouston[1] showed that no appreciable diminution in the number of fits took place when the dose of bromide exceeded 75 grs. in twenty-four hours.

[1] Clouston, *Journ. Ment. Sc.*, London, 1868.

The large doses sometimes prescribed—from 100 to 150 grs. daily—although no doubt suppressing the seizures for a time, induce other and more serious phenomena, namely, those of bromism already described. Moreover, the bromides have an accumulative action. Laudenheimer[1] has shown that an epileptic taking 10 grms. (150 grs.) of bromide salt daily for eight days, only excreted a total of 35 grms. or less than half the quantity ingested during that period. He also showed that no results followed its administration until an equilibrium was established between the intake and the output. This occurs on saturation of the body, and requires about 30 grs. of bromide to be given daily for three or four weeks. It is also largely dependent upon the amount of sodium chloride taken in food (p. 59).

Methods of Administration of the Bromides.—Most physicians have their own methods of prescribing the bromides in epilepsy. As already mentioned, large doses are not necessary, nor are they effectual in their results, but both the amount of the dose and the frequency and time of administration should be gauged by a study of individual cases. When I come to speak of the dietetic treatment in epilepsy, reference will be made to the deletion of salt, in the form of sodium chloride, from the dietary. This is an important modification in the treatment of epilepsy, as much smaller doses of the bromide may be prescribed. The potassium, sodium, strontium, and ammonium salts are the most usually administered. Each is of value, but the sodium salt is the most efficacious. If the bromides are prescribed in combination, the dose should not exceed 60 grs. in the twenty-four hours. The bromide of strontium is less useful, but may be given in smaller doses, or in combination with one of the other salts.

Combinations of the bromide salts, with other remedies, which may have some influence either upon the nervous or circulatory systems, have been from time to time recommended and are found useful in some cases. A combination of the bromides and digitalis has been found very satisfactory in cases of low arterial tension, irregular action of the heart, or failing compensation with valvular disease. Chloral hydrate may be

[1] Laudenheimer, quoted by Binswanger.

added with great advantage in cases of prolonged serial epilepsy, or of the status epilepticus. The bromides and the glycerophosphates form a valuable combination in weak or debilitated cases, more especially in young women with anæmia or neurasthenic symptoms. Bechterew recommends the conjunction of the bromides with adonis vernalis, and sometimes with codein. A combination of a bromide salt with borax has been of service where the bromides or borax, separately, have been of little use.

Bromide, Picrotoxin, and the Arseniate of Antimony.—(Gélineau's formula).[1]

Of the combinations of the bromides with other remedies, I have found Gélineau's formula the most useful and satisfactory. It is prescribed in the form of dragées, containing 1 grm. of the pot. brom., $\frac{1}{3}$ mgrm. of picrotoxin, and $\frac{1}{2}$ mgrm. of the arseniate of antimony. It has been used extensively in France, but does not seem to have been adopted to any extent in Britain. I have been in the habit of prescribing it during the past two years in those cases of epilepsy in which the pure salts of bromine are either not well borne or have been proved to be ineffective. In large doses, picrotoxin is a producer of convulsions, leading to spasms of a tetanic character with death in coma. In small doses, it is theoretically supposed to lessen the tendency to cerebral vaso-constriction, which is believed by some authors to be a fundamental factor in the causation of epileptic fits.

I have used the dragées in all forms of epilepsy with considerable success. My experience is that they are—

(1) A more successful remedy in most cases than a single bromide salt.

(2) Less likely to lead to troublesome symptoms, *e.g.* acne or bromism.

(3) More easy of administration, being especially convenient in either augmenting or diminishing the dose.

The method of prescribing them is simple—one dragée is taken either during or immediately after a meal, thrice daily for a week, an additional dragée being added weekly until the patient is taking four, five, or six per diem. I have found six

[1] Gélineau, "Traité des Épilepsies," Paris, 1901.

dragées a day usually sufficient to hold the fits in check, although, according to Gélineau, as many as ten or twelve daily may be taken. My experience of the remedy has not been sufficiently long to say definitely whether it is better in all cases than bromide alone, but, with two or three exceptions, it has been of great service in diminishing or even arresting fits in cases in which the bromides alone have been of no use.

Treatment by Drugs other than the Bromides.

In the days before the introduction of the salts of bromine in the treatment of epilepsy, many remedies were used, sometimes with marked success, as may be seen from the satisfactory results obtained by Herpin, Reynolds, and others. On account of the not infrequent failure of the bromides to arrest, or even to ameliorate epileptic attacks, it will be found necessary to prescribe some other medicinal remedy, and a large number have been from time to time advocated and employed. Perhaps the drug most frequently used as a substitute for, or as an adjuvant to, the bromides, more particularly in England, is *borax* (*sodium biborate*). Introduced by Gowers many years ago as an anti-spasmodic, it has met with considerable favour in cases where the bromides have been of little service. Unless in combination with a salt of bromine, I have not found it of particular use. It may be given in doses of from 10 to 30 grs. thrice daily, but is apt to induce troublesome gastro-intestinal symptoms. If continued over long periods, it may lead to cutaneous eruptions of a psoriasis-like character.

Belladonna was the chief anti-epileptic remedy of the pre-bromide days, and is still used in some cases with marked benefit when the bromides or other remedies have proved unsuccessful. It formed the chief remedy of Trousseau, Hufeland, Herpin, Reynolds, and others, and in the hands of the first named was mainly used in those cases complicated with nocturnal incontinence of urine. A combination of bromide and belladonna may be found useful in cases of otherwise intractable combined seizure types. It may be prescribed either in the liquid form, as the

tincture, in doses of 5 or 10 minims, or in the form of a pill made of the extract or pulvis belladonnæ. The *zinc salts* (oxide, valerianate, and lactate) are old established, and were occasionally successful, remedies in the hands of the French physicians. *Opium*, as a remedy for epilepsy, dates from classical times. It is now only used in the opium bromide method recommended by Flechsig.[1] My experience of the treatment has not been such as to encourage further trial. *Strychnine* has been recommended from time to time, and used with considerable success by some physicians. In doses of $\frac{1}{10}$ gr. daily, it may be continued over considerable periods. Its *modus operandi* is probably merely as a nerve tonic, although it may have some influence in strengthening the tone of the vaso-motor centres. Strychnine finds its most useful application in the treatment of nocturnal epilepsy, especially when there is reason to suppose that the blood pressure is materially lowered.

Monobromate of camphor, eosinate of sodium, chloretone, antipyrin, and other remedies have been tried, but without any special benefit. I have seen no particular advantage obtained by the use of bromipin, bromaline, or bromocarpine.

The Calcium Salts.

Recent examinations into the coagulation of the blood in epileptics have led to the suggestion that the salts of calcium may be of use in arresting or diminishing the frequency of epileptic seizures. As already shown, however, considerable discrepancy exists as to whether the blood in epilepsy coagulates more readily during a fit period. A more rapid coagulability during the four-and-twenty hours preceding a major seizure or serial attacks has been demonstrated (to my mind conclusively) by Dr. John Turner;[2] on the other hand, the observations of C. Besta[3] showed a diminished rate of coagulation—the fits apparently exerting no influence upon this. Donath[4] prescribed

[1] Flechsig, *Neurol. Centralbl.*, Leipzig, 1893, S. 229.
[2] John Turner, *Journ. Ment. Sc.*, London, 1908, October.
[3] Besta, *Riforma med.*, Roma, 1906, No. 43.
[4] Donath, *Epilepsia*, 1909, No. 1, p. 141.

chloride of calcium to a limited number of epileptics without materially affecting the number of the fits. I have myself prescribed the lactate of calcium in 10-gr. doses in combination with bromide of potassium in a few cases, with very satisfactory results, and Dr. Littlejohn[1] reports a case in which calcium lactate alone was prescribed with much benefit. It would therefore appear that the salts of calcium, which physiologically increase blood coagulation, are worthy of further study in the treatment of epilepsy. It is not unlikely that the special type of the disease most suitable for treatment by these salts has yet to be defined, as it is obvious that all fits are not associated with increased rate of blood coagulation.

Duration of Treatment.

The question as to how long bromide, or any other form of medicinal treatment, should be maintained, is not one upon which any rigid statement can be made. Some authorities maintain that treatment should be continuous for a period of at least two years after the last seizure; but our experience at the Queen Square Hospital points to a much longer period as necessary. Epileptics attend there for many years after the arrest of the fits, as they find that stopping the bromide, even for a short time, conduces to a return of giddiness, or of "sensations" reminding them of their previous attacks. If nine years' freedom from fits is to be a gauge of a "cure" of epilepsy, then withdrawing the bromide under a period of five years, in these in whom the fits are arrested, would be injudicious. On the other hand, many persons in whom the disease has been arrested after a year or two of bromide treatment, remain free from attacks without the aid of any medicinal remedy. The important practical point in this connection is, that those patients who take bromide well, and in whom the fits are thereby kept in subjection, ought to persevere with the remedy and not to stop it. It is just when the fits have been satisfactorily controlled that further treatment is of most use. The withdrawal of medicinal treatment in those

[1] Littlejohn, *Lancet*, London, 1909, vol. i. p. 1382.

in whom the fits have been arrested should be carried out gradually. Under no circumstances should bromides be withdrawn suddenly, after their prolonged use.

Miscellaneous Methods of Treatment.

It may be useful to refer to some of them in this place, as a case which has resisted one form of treatment may react, for a time, satisfactorily to another. It should, on the other hand, not be forgotten, that cases of this disease may respond favourably for a time to each and every change of treatment, medicinal or other, and even when active treatment is stopped. As the disease is characterised by spontaneous remissions in the frequency and severity of the seizures, a favourable result may occur, not on account of, but in spite of, therapeutic or other measures.

Of all the recent systems, that which seemed likely to be of most use was the introduction of the organic extracts in the treatment of this disease; but further experience with these preparations has been on the whole disappointing.

Organotherapy.—The administration of extract of the thyroid gland, or of iodothyrin, was at one time strongly advocated, more with a view to counteract the co-existent mental deterioration than as a subduer of convulsions. In a number of cases of confirmed epilepsy, in which preparations of the thyroid gland were given over considerable periods, no appreciable diminution was detected in the frequency of the seizures, and in only a limited number of cases, and for brief periods, was there any lessening of the co-existent dementia. My experience is that thyroid medication tends rather to increase the number of fits, and to produce at times irritability and want of control.

Preparations of the thymus gland act injuriously in epilepties by increasing the number of the seizures. Cerebrin has not been found to be of any value.

Serotherapy.—The treatment of epilepsy by the injections of blood serum, either from another epileptic, or by reinjection of the blood serum into the same epileptic, was introduced by

Ceni,[1] who believed that a soluble epileptogenous poison existed in the blood serum in quantities varying with the severity of the disease. Ceni's view differed from that held by most supporters of the autotoxic theory of convulsions, in that the amount of poison circulating in the blood did not change in the different stages of the disease. As the result of a number of observations, Ceni concluded that there exists in the blood of epileptics a soluble latent biochemical substance, an autocytotoxin, which has an influence over the elaboration of epileptogenic toxic agents, and produces its results after repeated injections. The records of these observations show that in some cases a beneficial, and in others an unfavourable, result followed the injection of the blood serum of epileptics into other epileptics.

The practical utility of this method in the treatment of epileptic fits, even in the hands of its originator, has not been sufficiently satisfactory to render its application general; while later investigations, more particularly those of Sala and Rossi,[2] Gerharz,[3] and others, failed to confirm Ceni's results, or to establish any benefit at all from the injection of the blood serum of epileptics into others subject to epilepsy.

Special Treatment.

To arrest the Fit.—The first consideration may be given to the question as to whether it is possible to arrest an attack, once the warning has commenced. Many methods have been suggested for this purpose, some of which are of old standing and date from the time of Galen. The attacks in which abortive measures are likely to be successful are those commencing with a peripheral aura. The common method of encircling the wrist, for example, with a ligature or tape, and making traction upon it as soon as the aura is felt in the hand, is well known. As great force is sometimes required to arrest the attack, a strap is preferable to a tape or ligature. Sometimes the patient alone is

[1] Ceni, *Riv. Sper. di freniat.*, Reggio-Emilia, 1901, p. 344; and *Neurol. Centralbl.*, Leipzig, 1903, S. 388.

[2] Sala and Rossi, *Neurol. Centralbl.*, Leipzig, 1903, S. 852.

[3] Gerharz, *Neurol. Centralbl.*, Leipzig, 1904, S. 835.

unable to produce sufficient compression, and requires the assistance of a second person. A circular blister was suggested by Buzzard,[1] in order to induce a more permanent effect, sometimes with advantage.

Forced extension or movement in the direction opposite to the warning sensation may be efficacious, when compression alone is unsatisfactory. According to Herpin,[2] the most effectual means of arresting such attacks is a combination of circular compression and forced movement in the opposite direction. Friction, or rubbing the extremity of the limb where the sensation starts, has also been of use; and I have known a patient to bite the finger in which the aura commenced sometimes with a successful issue.

Abortive means are less satisfactory in cases with a visceral aura. Strong pressure by the hands over the epigastrium is resorted to by some epileptics, while others prefer to drink cold water; swallowing a few drops of ether has also occasionally resulted in arresting an attack. Inhalations of ammonia have been used successfully. I recall a case in which a seton inserted over the epigastrium was employed with advantage in diminishing the frequency of the attacks. Other patients refer to a method of auto-suggestion, bringing to bear a strong determination to overcome the attack—a method which undoubtedly has been followed by success in some cases.

The inhalation of nitrite of amyl is a method of arrest, more especially valuable in fits with cephalic warning, and particularly in those with olfactory sensations (Gowers). Pierce Clark has advocated this remedy as a satisfactory means of aborting fits.

Status Epilepticus.—It has been shown that a gradually increasing number of seizures is the usual sign of the onset of the status epilepticus or of prolonged serial outbursts. With this warning the dose of bromide should be increased even up to double the quantity, and chloral (10 to 15 grs.) should be added, and the mixture repeated every four hours. Clark recommends the addition of a solution of morphia to the draught.

[1] Buzzard, *Brain*, London, vol. iii. p. 554.

[2] Herpin, "Acces Incomplet de l'Epilepsie," Paris, 1867, p. 29.

Should the fits be recurring with great frequency and severity, no remedy is of greater benefit than the inhalation of chloroform, given up to complete anæsthesia. On the other hand, in less severe types of status, or in serial epilepsy, a combination of a bromide salt (20 grs.) and chloral hydrate (10 grs.) may be repeated frequently (about every two or three hours) for a time with benefit, especially in the latter condition.

The bromides given alone are of little avail; but within recent years their hypodermic administration in sterile solutions of not more than 10 per cent. (Clark) has been recommended; these may be repeated until 60 or 100 grs. have been injected.

Injection of the bromides by means of *lumbar puncture*[1] has also been advised, in sterile solutions of 30 grs. to the ounce, 10 or 15 c.c. of the cerebro-spinal being withdrawn before 10 c.c. of the bromide solution are injected.

The hydrobromate of hyoscine has also been used occasionally with success ($\frac{1}{75}$ to $\frac{1}{100}$ gr. hypodermically).

In the post-convulsive stuporous stage of status epilepticus the treatment is that of acute amentia — strychnine, digitalis, and alcohol, if there is much cardiac debility. Abundance of nutritious food and careful nursing are essential features in the general management of this period.

Acute Amentia.—During the after-stage of exhaustion following upon ordinary seizures, no special treatment is necessary, as the stage passes into that of sleep, from which the patient spontaneously recovers.

In the acute amentias following serial or status outbursts, on the other hand, great care and attention is required, the patient having to be nursed as one suffering from acute illness. It is during this stage that death may occur, a circumstance which is as frequently attributable to want of attention, as to the clinical condition. During the few days of stupor, abundant and nourishing liquid diet, in the form of milk, eggs, and custards, should be frequently given. If the patient is unable to swallow, nourishment should be administered in the form of nutrient enemata. Hypodermic injections of strychnine (liq. strych. 5

[1] Morton, *Trans. Nat. Society*, U.S.A., vol. iii. p. 42.

minims or stryeh. sulp. $\frac{1}{30}$ gr.) may require to be frequently administered.

If neeessary, aleohol may be given in eonsiderable doses, and the aetion of the heart steadied and maintained by digitalis, or strophanthus. Later on, during the delusional stage, general attention and eare is all that is usually needed, while later tonies may be preseribed with advantage.

Acute Mania.—As already deseribed, this form of exeitement, whether oeeurring as a post-paroxysmal phenomenon or as a psyehieal equivalent, is eharaeterised by the suddenness of its onset, the intensity and violenee of its manifestations, and the shortness of its duration, extending usually over a few hours. All that is therefore required lies in proteeting the patient, and those attending him, from the effeets of the violenee and exeitement. For this purpose resort may be had to the serviees of attendants, or, if a drug be considered advisible, I know of none more safe to administer, or more speedy, eertain, and satisfaetory in its aetion, than the hydrobromate of hyoseine in doses of $\frac{1}{75}$ to $\frac{1}{50}$ gr., injeeted hypodermieally. One injeetion is usually suffieient to induee quiet and repose for a period of several hours.

Dietetic Treatment.

In all eases of reeent epilepsy, whatever may be the medieinal treatment adopted, some modifieation of the diet from that of a person in normal health is desirable.

In the treatment of this disease the primary objeet is to keep the dose of the bromide salts as small as possible, beeause, owing to the protraeted nature of the malady, a prolonged eourse of bromide medieation is necessary, partieularly in those cases in whieh treatment is most effeetive. Therefore any dietetie modifieation whieh will permit of this being aeeomplished is of advantage.

As already pointed out in the first leeture, many eauses have been assigned as the immediate exeitant of epileptie seizures, but the only eertain faetor is the unstable condition and proneness to "diseharge" of the eortical nerve cells in predisposed persons.

In any attempt to formulate the principle which ought to guide the physician in considering the dietetic treatment, there ought to be kept in mind that nervous energy has its source chiefly in the albuminous and nitrogenised principles of food-stuffs.

Merson [1] treated a number of epileptics upon farinaceous and nitrogenous diet alternately over a stated period, and noted the number of the seizures. The results of the treatment were not such as to justify the conclusion that either kind of diet possessed any decided advantage over the other in the treatment of epilepsy. The observations of Alt [2] upon the several forms of diet in epilepsy led him to the conclusion that a diet without meat was the most satisfactory, but that neither a milk diet alone, nor a vegetable diet, was as beneficial as their combination.

It was an old speculation of Hughlings Jackson that the part played by salts and minerals should be investigated in their relation to the building up of structure and the development of function. As the chlorides and the bromides were strikingly homologous in their chemical and physical properties, he suggested that the efficacy of the bromide salt might be due to their replacing with greater energy the more common chloride salts in the blood and tissues of the body.

Salt-starvation.—The practical application of this suggestion, however, was not carried out in the treatment of epilepsy until Toulouse and Richet [3] recommended a diet in which the total quantity of sodium chloride per diem was limited to one or two grammes. It was thought that by diminishing the quantity of the ingested chlorides, mainly in the form of sodium chloride or common salt, the bromides might be administered in smaller doses, and the risks of bromism thereby lessened.

Hoppe [4] has shown that one-third of the chlorine of the blood serum has to be replaced by an equivalent amount of bromine before any therapeutic result is obtained. When more than this is replaced, bromide intoxication may occur.

[1] Merson, *West Riding Asylum Reports*, 1875, vol. v.
[2] Alt, *Ztschr. f. klin. Med.*, Berlin, vol. liii.
[3] Toulouse and Richet, *Acad. des Sciences*, Paris, tome xi. p. 20.
[4] Hoppe, *Neurol. Centralbl.*, Leipzig, 1906, S. 993.

When less chloride is ingested, saturation takes place sooner. It has been shown that with a diet free from salt, saturation takes place in from three to four days. This method of "salt-starvation," in the treatment of epilepsy has been extensively tested with varying results.

In some cases "salt-starvation" has proved a useful adjuvant to bromide medication, while in others little benefit has resulted. Other observers have shown that the dietary is of especial value in cases requiring large doses of the bromides, and in those which show a ready tendency to bromide intoxication. My own experience of the method has been such that, when used in combination with a purin-free dietary, very substantial benefit is derived, and relatively small doses of the bromides may be prescribed.

Purin-free Dietary.—A "purin-free" diet is made up of those food-stuffs in which the "purin" or alloxur bodies are absent, or present only in such small quantities as to be negligible.

Purin bodies exist in all forms of meat extract, in both the white and the red meats, commonly used as food. They are present in large quantities in such substances as sweetbread, liver, and beefsteak. They are not present in milk, eggs, bread, butter, cheese, the farinacea, most fruits, some vegetables, and honey. They exist to only a moderate degree in most forms of fish, peas, beans, lentils, tea, coffee, and oatmeal. I have drawn out a list of the common foods with their purin value from the analysis of Dr. Walker Hall (see Appendix); and I give a dietary which will be found of use to those desirous of giving the treatment a trial.

Diet Table.

Milk (fresh, soured, butter-milk, or whey).
Eggs (boiled, poached, scrambled, or raw).
White bread and butter, cheese, macaroni, rice, tapioca, semolina, vermicelli.
Suet puddings with currants, jam and treacle, apple dumplings.
Pastries, jellies, pancakes, tea-cakes.
All vegetables (except peas, beans, lentils).
All fruit; sweetmeats are harmless.
Of beverages, weak infusion of China tea is the best.

I have used this diet, or a modification of it, containing, aeeording to the needs of individual cases, a small portion of fish, either once daily or three times a week, for several years, in eonjunetion with the bromides or Gélineau's dragées. The results have been such that I am led to advise it in all cases of reeent epilepsy.

1. In eases in whieh the bromides alone have been of little or no use, the adoption of the purin-free saltless diet has at onee led to material improvement.

2. By its aid the dose of the bromides has been largely redueed, as it is in eases refraetory to relief by bromides that some physicians increase the salts to such an extent that bromism is brought about and maintained.

3. If properly supervised, symptoms of bromism need never appear.

4. If the patient show any signs of loss of weight, the addition of eream or eod-liver oil is usually sufficient. If not sufficient, it is advisable to permit fish or even a little lamb or mutton.

Confirmed Epilepsy.

In cases of eonfirmed epilepsy with mental deterioration, the use of the bromides is of relatively little value. Aecording to Féré, the bromides are only of use when the dose is so large as to produee toxie effeets. Their continued administration in these cases does, however, produee a diminution in the number, and perhaps in the severity, of the seizures. With a view to test the efficieney or otherwise of a 30-gr. dose of bromide salt administered every evening to a number of eonfirmed epilepties in an institution, the dose was stopped for one month, and the total number of fits with or without bromide was compared. During this period the fits were increased in frequeney by about one-third (278 to 402).

My experienee of the treatment of eonfirmed epilepsy is that the administration of the bromides is of little real service. It was long ago pointed out by Esquirol[1] that any ehange of

[1] Esquirol, "des Maladies Mental," Paris, 1838, tomo i.

treatment was temporarily beneficial. Bringing epileptics under the generally favourable hygienic surroundings of a hospital will often conduce to material temporary improvement without the aid of any medicinal remedies. As with medicinal treatment, so with dietetic modifications, I have not seen any material benefit obtained in cases of confirmed epilepsy by aid of the salt-free or purin-free diets. The majority of the cases feel better when on the purin-free diet, but there is no marked improvement in the frequency or severity of the seizures, except perhaps in those cases in which the fits occur in series. These observations would bear out what has been constantly noted in the treatment of epilepsy, namely, that in the confirmed disease, with mental impairment, little, if any, benefit is derived from treatment, either medicinal or dietetic. In other words, those cases in which the disease has become established upon an organic foundation require to be lodged preferably in an institution for epileptics, where they may be prescribed (1) regular and congenial employment, (2) a judicious alteration of work and play, and (3) a suitably arranged and simple mode of life, with avoidance of excitement and abstinence from alcoholic liquors.

APPENDIX.

Purin Value in Grains per lb. of the common Food-Stuffs (Walker Hall).

Sweetbread	70·4	Mutton	6·7
Liver	19·2	Plaice	5·5
Beefsteak	14·4	Cod	4·0
Sirloin	9·1	Beans, lentils	4·1
Chicken	9·0	Oatmeal	3·4
Pork	8·4	Asparagus	1·5
Veal	8·1	Coffee	1·7
Salmon	8·1	Ceylon tea	1·2
Ham	8·0	China tea	0·75
Halibut	7·1	Onions	0·06

Purin-Free Foods.

Nuts, chestnuts, raisins, dates.
Cheese, macaroni, white bread, potatoes.
The farinacea.
Milk, cream, butter, fats, eggs.
Apples, grapes, figs.
Honey.
Olive oil.

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